CARBON DIOXIDE EFFECTS UNDER CONDITIONS OF RAISED ENVIRONMENTAL PRESSURE

by

K. E. Schaefer, M.D.

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Reviewed and Approved by:

Charles F. Gell

Charles F. Gell, M.D., D.Sc.(Med.)
SCIENTIFIC DIRECTOR
NavSubMedRschLab

Approved and Released by:

R. L. Sphar

R. L. Sphar, CDR, MC, USN
COMMANDING OFFICER
NavSubMedRschLab

SUMMARY PAGE

THE PROBLEM

Carbon dioxide plays an important role in the physiology of the high-pressure environment, since the increased breathing resistance results in carbon dioxide retention. This represents one of the major limitations in diving operations.

FINDINGS

The extent to which CO_2 retention affects or contributes to the known depth limits in diving operations has been determined in such areas as: breathhold diving, scuba and helmet diving, submarine escape (buoyant), He-O_2 saturation diving, and shallow habitat air diving. Moreover, CO_2 retention during exertion at high ambient pressures is discussed in considerable detail as well as the role of CO_2 in inert gas narcosis. Finally, data on adaptation to CO_2 in breathhold and scuba divers are summarized.

APPLICATION

This information is of interest to Submarine and Diving Medical Officers and scientists interested in Underwater Physiology and Medicine.

ADMINISTRATIVE INFORMATION

This investigation was conducted as a part of the Bureau of Medicine and Surgery Research Work Unit M4306.02-7060 - "Regulation of Respiration and Circulation in Rest and Exercise in Naval Diving Operations". The present report is No. 6 on this work unit. The manuscript was submitted for review on 14 August 1974, approved for publication on 26 December 1974 and designated as Naval Submarine Medical Research Laboratory Report No. 804.

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ABSTRACT

Exposure to increased pressure, such as that encountered under water, affects the mechanics of respiration and in particular, the behavior of the respiratory gases. Carbon dioxide plays a major role in the physiology of the high-pressure environment, since the increased breathing resistance easily leads to carbon dioxide retention. The latter has been observed frequently in scuba and helmet diving.

Pulmonary gas exchange in breathhold diving is influenced by the compression and decompression events resulting in a reversed carbon dioxide gradient during descent. The alveolar carbon dioxide level can be controlled during the ascent from depth by controlling the speed of ascent, a fact which is of significance to both breathhold diving and the buoyant ascents of submarine escape.

Investigations of pulmonary gas exchange in rest and exercise during exposure to high pressure while breathing helium-oxygen gas mixtures have demonstrated the existence of respiratory limitations and associated CO_2 retention when divers are performing heavy, exhaustive work.

In shallow habitat air diving using combinations of air and normoxic nitrogen-oxygen breathing mixtures, evidence for the development of slight respiratory acidosis and CO_2 retention has been obtained.

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INTRODUCTION

Exposure to the increased pressure encountered under water affects the mechanics of respiration and in particular the behavior of respiratory gases. Carbon dioxide plays a major role in the physiology of the high-pressure environment since the increased breathing resistance easily leads to carbon dioxide retention. The latter has been observed frequently in scuba and helmet diving.

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BREATHHOLD DIVING

Pulmonary gas exchange

Pulmonary gas exchange during breathhold diving has been extensively studied in tank instructors at the 118 ft Submarine Escape Training Tank, U.S. Naval Submarine Base New London, in dives to 90 ft (3.7 ATA) (Schaefer 1955; Schaefer & Carey 1962). During descent, the ambient pressures increase rapidly resulting in a compression of chest and lungs. It was found that the carbon dioxide tension in the lungs rose quickly above the venous carbon dioxide tension and a reversed carbon dioxide gradient developed. At 90 ft, (3.7 ATA) approximately 50% of the pre-dive carbon dioxide content of the lungs had disappeared and was taken up by the blood and tissues. The influx of carbon dioxide into the lungs during ascent appeared to be rather slow and it was found possible to control the alveolar carbon dioxide level by controlling the speed of ascent. If the ascent was fast, the alveolar carbon dioxide tension attained on reaching the surface was low, being between 30 and 35 mm Hg, but if the ascent was slow the alveolar carbon dioxide tension rose to 40 to 45 mm Hg. The alveolar oxygen tension rose from control levels of 100 mm Hg to 300 mm Hg at 90 ft (3.7 ATA) and fell on ascent rapidly during the last 10 ft (1.3 ATA) to values as low as 25 to 30 mm Hg.

The disappearance of carbon dioxide from the lungs during dives, together

with the oxygen utilization and mechanical compression of the thorax as the subject descends, produces a progressive shrinkage of the total chest volume.

A diver at 90 ft (3.7 ATA) is somewhat protected inasmuch as the carbon dioxide tension does not rise to dangerous levels and the oxygen tension remains rather high. Under these conditions, breathholding time is considerably prolonged. However, during the last part of ascent or just at the moment the diver reaches the surface, available oxygen may become so depleted as to produce hypoxia. Thus, the alveolar oxygen falls to a very low level at the end of ascent from dives to 90 ft (3.7 ATA). As an example, one instructor-subject became confused in the initial moments after reaching the surface during which time he gave an alveolar sample, but he quickly recovered after the first breath. His alveolar oxygen concentration was 3.5% (PA_{O_2} =28 mm/Hg). With a very low-oxygen content and a normal or below normal carbon dioxide concentration, the nitrogen content of the alveolar air at the end of the dive is markedly increased, 89% compared with a normal 79%.

These findings can be explained by the difference in rate of elimination of nitrogen compared with carbon dioxide. Nitrogen, carbon dioxide and oxygen gas tensions in the lungs are increased fourfold at a depth of 99 ft (4 ATA). Under these circumstances, nitrogen as well as carbon dioxide and oxygen, diffuse from the lungs into the blood. Nitrogen follows the law of solubility of gases in liquids in relation to partial pressure. In carbon dioxide uptake by the blood and tissues, three factors play a role, namely, solubility in liquids, chemical combination with an alkaline buffer and carbon dioxide transfer between the plasma and the cell system. The reservoir for absorbing carbon dioxide appears much larger than that for nitrogen. During ascent, which represents a form of rapid decompression, nitrogen is rapidly released from a small store under high pressure, whereas it takes longer to release carbon dioxide from a large store. On the basis of theoretical calculations, DuBois (1955) has predicted such changes in pulmonary gas exchange during diving, and Bjurstedt and Hesser (1956) confirmed the existence of a reversed carbon dioxide gradient during compression in dog experiments simulating skin dives to 130 ft (4.9 ATA). Lanphier and Rahn (1963) investigated gas exchange during simulated breathhold dives in man and obtained similar results, which were later confirmed in field studies on diving pattern and alveolar gas exchange in Korean diving women (Hong, Rahn, Kang, Song & Kang 1963). The most comprehensive information on the physiology of breathhold diving is contained in the symposium arranged by Rahn and Yokoyama (1965).

The influx of carbon dioxide into the lungs during ascent is regulated at least in part, by the speed of ascent. Table 1 shows the alveolar carbon dioxide and oxygen tensions measured after surfacing from dives in which three different rates of ascent were used. With faster ascents the alveolar carbon dioxide tensions attained on reaching the surface decreased.

TABLE 1

End-dive $P_{A_{CO_2}}$ and $P_{A_{O_2}}$ values following descent to 90 ft and ascent with different speeds

Dives to 90 ft	*Alveolar gas tensions*	
speed of ascent	P_{CO_2} mm Hg	P_{O_2} mm Hg
1·9 ft/sec	45·7 ± 4·1 (12)	34·8 ± 5·7 (12)
2·3 ft/sec	37·3 ± 1·2 (3)	34·7 ± 4·2 (3)
3·5 ft/sec	31·5 ± 1·3 (3)	27·3 ± 3·1 (3)
Breath-holding at surface	52·4 ± 2·4 (12)	64·8 ± 13·4 (12)

Number of dives indicated in parentheses.

Breath-holding at the surface was carried out by the same subjects who performed the dives to 90 ft and ascended at an average speed of 1·9 ft/sec. Breath-holding time of 1·5 min corresponded with the average time of breath-hold dives.

Measurements of blood gases, lactic acid, respiration and metabolism were made on four subjects before and after diving to 90 ft (3.7 ATA). The carbon dioxide content of blood rose very slightly while the oxygen content fell during the dive. The lactic acid content in the oxygen increased over five-fold in a sample that was taken one minute after the dive. The lactic acid decreased to a level slightly above normal within five minutes. On more frequently collected venous samples, peak lactic acid concentrations were also measured after three minutes of recovery following the dive. The one-minute values were consistently lower than the three-minute values. This corresponds with similar findings obtained in pearl divers by Scholander, Hammel, Le Messurier, Hemmingsen and Carey (1962). The delayed but large rise in lactic acid, found during the recovery phase in man, is quite similar to that observed in the seal and might, according to Scholander et al (1962), also be interpreted as an indication of reduced muscle-blood flow during the dive.

Respiration was increased three-fold during the first minute after the dive and returned to normal levels within 15 minutes. The excess oxygen up-take above the control level after the dive was limited to four minutes and averaged 1400 ml. in four subjects. This indicates that the oxygen debt occurring during the dive of one and one-half minutes is in the order of 1400 ml. The excess carbon dioxide exhalation within the first four minutes after diving averaged 900 ml. (Schaefer 1955).

To investigate pulmonary and circulatory adjustments which determine limits of depths in breathhold diving, data on pulmonary gas exchange were collected in open-sea breathhold dives to depths of 217.5 and 225 ft on diver Croft who made a world record dive to 240 ft on 12 August 1968 (Schaefer et al

1968). Thoracic blood volume displacements were measured at depths of 25, 50, 90 and 130 feet, by use of the impedance plethysmograph. The open sea dives were carried out with an average speed of descent of 3.95 ft/sec. and an average rate of ascent of 3.50 ft/sec.

End dive alveolar oxygen tensions did not fall below 36 mm Hg, while alveolar carbon dioxide tension did not rise above 40 mm Hg except in one case (Figure 1). These findings indicate that for diver Croft, who has an unusually large lung capacity, neither hypoxia nor hypercapnia determined the depth limits under those conditions. At depths of 90 and 130 feet, blood was forced into the thorax, amounting to 1047 and 850 milliliters, respectively.

Since blood-shifts into the thorax have been shown to play a significant role in allowing the breathhold diver to go to a deeper depth than could be predicted from measurements of total lung capacity and residual volume, studies were carried out in the wet and dry chamber to determine whether the blood shifts are caused by hydrostatic pressure only. Measurements of transthoracic resistance (impedance plethysmograph) made during simulated breathhold dives to 25, 50 and 90 feet in the wet and dry chamber demonstrated that blood shifts into the thorax during breathholding are caused by hydrostatic pressure, since no change in thoracic resistance occurred during breathhold dives in the dry chamber (Schaefer et al 1972).

Paulev (1969) carried out extensive investigations of respiratory and cardiovascular effects of breathholding. He found that breathholding with an intrapulmonic pressure of 20-40 mm Hg (Valsalva maneuver) produced a drastic reduction of limb blood-flow, if the arterial blood pressure fell appreciably. On the basis of his experiments and studies on 199 persons, Paulev concluded that loss of consciousness with the risk of drowning may be caused by CO_2 narcosis in combination with the well-known hypoxia during ascent from deep breathhold dives. Hong et al (1971) investigated the time course of alveolar gas pressures and alveolar O_2 and CO_2 exchange together with changes in cardiac output and blood pressure during prolonged breathholding with air. During four minutes of breathholding, the lung supplied 700 ml. of O_2 into the blood while it gained only 160 ml. of CO_2 from the blood indicating a significant retention of CO_2. The CO_2 increased during the first 30 seconds and leveled off at about 50 mm Hg. $P_{A_{O_2}}$ fell

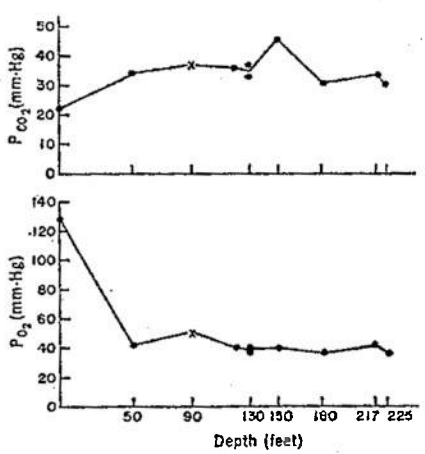

Fig. 1. End-dive alveolar gas tension obtained after rapid ascent at an average rate of 1.2 m/sec from various depths. Control values (left most): alveolar gas tensions after rapid exhalation following maximal inhalation. X tank dive; circle, open-sea dive. From Schaefer et al (1968) with permission of the publisher.

continuously and reached approximately 30 mm Hg. at the end of breathholding at 4 minutes.

Cardiac output did not change significantly. These observations on alveolar gas exchange during breathholding confirm and extend the findings obtained previously during one-minute breathholds by Craig and Harley (1968) and breathholds longer than one minute by Hong et al (1970) and Tibes and Stegemann (1969).

Craig and Medd (1968) measured oxygen consumption and CO_2 production during single and repetitive dives to 5 and 10 m. and the results of these experiments were compared to those from underwater swimming and land exercise with breathholding. CO_2 retention observed during the dive was greater after the deeper dives. It was concluded that part of the CO_2 retention was related to the effect of increased ambient pressure and lung compression. Water immersion itself must play a role, since the P_{ACO_2} at the end of the underwater swim was not as high as it was after the land exercise with breathholding.

SUBMARINE ESCAPE (FREE AND BUOYANT ASCENT)

Buoyant ascent (aided by an inflated life jacket) has been successfully carried out from depths of 300 ft (10 ATA) at an ascent rate of 340 ft/min. (10.3 ATS) without respiratory distress (Bond, Workman & Mazzone 1960). Evaluation of alveolar gas exchange data obtained during buoyant ascent from 90 ft (3.7 ATA) have shown that the alveolar carbon

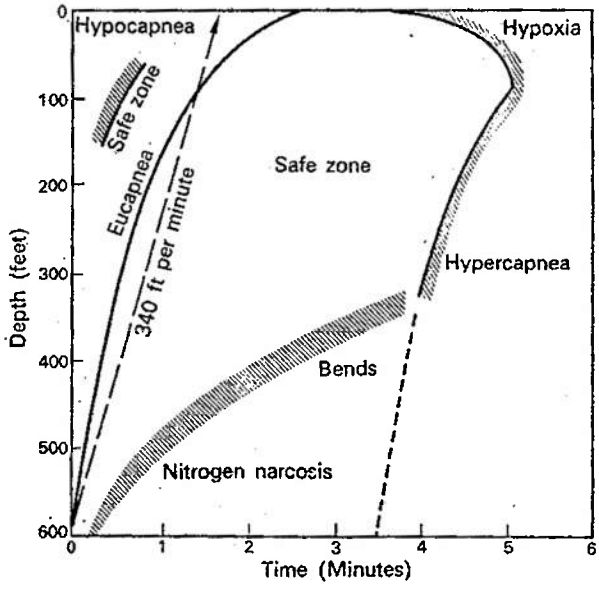

Fig. 2. Depth-time curve for buoyant ascent (submarine escape) from 600 ft (19 ATA) From DuBois, Bond and Schaefer (1963) with permission of the publisher

dioxide tension can be kept at normal levels at the average ascent rates used (DuBois, Bond & Schaefer 1963). A series of successful ascents made in 1965 by submariners of the Royal Navy from depths down to 500 ft (16 ATA) have been described (Elliott 1966). A theoretical depth-time curve for buoyant ascent was established, according to which it appears possible to make these ascents from 600 ft (19 ATA) without being endangered by carbon dioxide narcosis, nitrogen narcosis or anoxia (Figure 2).

Escape from depths of 600 feet has been accomplished by the Royal Navy in open sea trials with a pressurization time of 20 sec., bottom time of 1.5 sec. and a free ascent rate of 8.5 ft/sec. to the surface (Donald 1970).

It has been proposed that the depth for submarine escape could be considerably extended by using a hyperbaric suit which enables the submariner to attain an increasing ascent rate and to maintain an overpressure for a certain period on the surface which would be equivalent to a decompression stop at 10-20 feet. (Eaton and Hempleman 1971). It has also been suggested to substitute carbon tetrafluoride for nitrogen as an inert gas diluent in submarine escape procedures which would double the depth capability (Gait and Miller 1973).

Since carbon tetrafluoride is a more slowly saturating inert gas, oversaturation in any critical tissue can be avoided during the short exposure periods required during submarine escape procedures.

These authors found in LD_{50} studies in mice exposed for one minute to high pressure of carbon tetrafluoride or nitrogen with sufficient oxygen available in the breathing atmosphere, a 35% greater depth for carbon tetrafluoride.

SELF-CONTAINED DIVING

Several cases of unexplained loss of consciousness were observed with the use of oxygen closed-circuit diving equipment in which canisters are employed for carbon dioxide removal (Barlow & MacIntosh 1944). Carbon dioxide intoxication was implicated as the most likely cause of the "shallow-water blackout". Additional investigations demonstrated that it is possible in most subjects to produce a marked carbon dioxide intoxication without severe respiratory dyspnea. In other words, the depressant effects of carbon dioxide can manifest themselves without the warning sign of a strong respiratory stimulation. Predominance of depressing carbon dioxide effects over respiratory stimulation are facilitated by physical exertion and high-oxygen tensions. However, Miles (1957) has suggested that shallow-water blackout might be caused by sudden increases in oxygen tensions leading to oxygen syncope. When an open- or a closed-circuit scuba is used at great depth, the direct effect of pressure produces an increased density of the breathing mixture resulting in increased resistance to breathing. Under these conditions, the work of breathing is increased by the resistance developed in the breathing apparatus and in the airways of the diver (Marshall, Lanphier & DuBois 1956). Pulmonary resistance at 100 ft (4 ATA) increases two-fold compared with values at sea level (Mead 1955).

Although the underwater breathing equipment has been considerably improved over the years, divers still experience difficulties during deep dives. Such problems were reviewed in a recent workshop on "Respiratory Limitations of Underwater Breathing Equipment" (Bradley 1974).

Serious respiratory limitations were encountered with different types of underwater breathing equipment during the SeaLab III experiment, in the 1010-foot open-sea dive and in a 640-foot open-sea dive using neon. In the SeaLab III experiment, the divers were not thermally supported and were working from a depth of

620 ft breathing helium-oxygen with a Mark IX UBA equipment, which did not allow them to get enough gas. They also were affected by severe cold. One of the divers died in this experiment due to hypercapnia and cold effects. The other diver was unable to complete the rescue because of his own respiratory difficulties (Bradley 1974). It should be pointed out that carbon dioxide accumulation in the body has severe effects on thermoregulation. When higher CO_2 concentrations are reached, heat production is impaired while heat loss is increased (Schaefer 1974). In the 1010-foot open sea dive, the divers were thermally supported but encountered respiratory difficulties even during minimal exercise using a Mark II DDS, and a Kirby-Morgan Band Mask. Laboratory tests carried out with this mask showed that the mechanical work required for breathing dense gas with this equipment was extremely demanding (Storrie 1974). In the 640-foot open sea dives neon-oxygen mixtures were used as breathing gases with the Kirby-Morgan Band Mask. During the working dives at this depth, the divers became hypercarbic, but did not recognize this condition. The inspired oxygen was rather high (1.6 ATA) which might have contributed to the CO_2 retention (Hamilton 1974). Physiological effects of underwater breathing equipment on ventilation and gas exchange have to be taken into consideration.

During head-out immersion in the erect position, cardiac output increases by more than 30 percent and perfusion of muscle tissues (anterior tibial) rises more than 100 percent (Lundgren 1974). The increase in circulation enhances nitrogen elimination and could, in conjunction with oxygen breathing, result in pulmonary atelectasis. Furthermore, recent experiments at Duke University have demonstrated that the resting arterial P_{CO_2} rises with the ambient hydrostatic pressure, on the order of 0.5 mm Hg per atmosphere of ambient pressure increase (Saltzman et al 1971 and Saltzman 1974).

In dogs exposed to high pressure of helium-oxygen, a change in the electrochemical potential difference for bicarbonate between plasma and the central spinal fluid (CSF) was found which is indicative of an increase in the concentration of CSF bicarbonate. Saltzman (1974) interpreted these findings as suggesting the development of central alkalosis with increasing hydrostatic pressure, perhaps due to alteration of the blood brain barrier for bicarbonate. These findings have a number of implications for diving physiology. The direct effect of the hydrostatic pressure raising the arterial P_{CO_2} independent of respiratory limitations brings up questions about a steady state arterial P_{CO_2} of a diver at 1000 or 2000 feet. What would be a "normal value" under these conditions when there is no respiratory limitation? This is of particular importance for the evaluation of underwater breathing equipment using physiological tests.

To verify the observations of Saltzman et al (1971) of a positive correlation between $PaCO_2$ and ambient pressure made in human subjects, Kerem and Salzano (1974) studied the effects of 18 hours of exposure of unanesthetized dogs to a helium-oxygen mixture (3.4% or 16.6% helium) at an

ambient pressure of 6.45 ATA. The gas density was only slightly raised (to 1.17) relative to that of air at 1 ATA and the oxygen concentration remained at normoxic levels. Arterial and cerebrospinal fluid (CSF) P_{CO_2} increased 5 and 4 mm Hg, respectively, CSF bicarbonate rose 2 meq/L, CSF pH was not changed. Arterial pH decreased .04 units, the plasma standard bicarbonate did not change. These findings confirmed the observations of Saltzman et al in humans and indicate that increased gas density and elevated inspired oxygen concentrations do not seem to play a role in the hydrostatic effect on $PaCO_2$.

Physiological testing methods have been proposed for underwater breathing apparatus. At the U.S. Navy Experimental Diving Unit (EDU) tests have been performed on breathing equipment at different working depths in which a limit of the arterial blood P_{CO_2} was set at 50 mm Hg. If higher levels of CO_2 were reached, the equipment was considered unsafe (Strauss, Wright et al 1972).

Another limit of 11 kg-m/min. for respiratory work has been used which is based on clinical studies showing that patients with chronic pulmonary disease frequently develop "respiratory failure" if their ventilatory work exceeds this level.

Both of these limits are arbitrarily defined as a first measure to set physiological criteria for the testing of underwater breathing equipment and need further studies to confirm their validity.

HELMETED DIVING

In deep diving, in which the conventional suit and helmet are used, a large amount of air must be ventilated to prevent the accumulation of carbon dioxide. Often this may not be fully accomplished. Moreover, at great depths, breathing resistance becomes very marked and may easily lead to carbon dioxide retention. Lanphier found that a considerable number of experienced helmeted divers at the U.S. Navy Experimental Diving Unit experienced carbon dioxide retention during underwater work (Lanphier 1955 a, b). The respiratory minute volume declined during working dives to moderate depths using oxygen-nitrogen mixtures. The degree of retention of carbon dioxide was related to the ventilatory response to carbon dioxide (Lanphier 1956). Those with a high tolerance to carbon dioxide retained more carbon dioxide. When breathing resistance was reduced by the use of helium-oxygen mixtures, the carbon dioxide retention was small or absent. A lowered sensitivity to carbon dioxide was frequently found in these divers (Lanphier & Morin 1961).

More recent studies carried out in the Swedish Navy by Muren and Wulff (1972) demonstrated that CO_2 retention found in the diver's helmet during heavy work is mainly caused by an unequal distribution of CO_2 and can be greatly alleviated by the insertion of an air diversion tube.

Simulating the flow rates used in practical diving, 60 and 40 lit/min. P_{CO_2} levels measured at three different locations, helmet, throat and suit

were 12, 19 and 23 mm Hg with 40 lit/min. ventilation during work.

Considering a P_{CO_2} of 20 mm Hg as a critical limit, an attempt was made to improve the unequal distribution of CO_2 in the helmet.

A number of symptoms occurred during heavy work, which implicated CO_2 retention as a casual factor.

The divers often reported a sudden lack of strength, occasionally associated with dizziness. It was observed that the normal reaction of an experienced diver under those conditions is to relax and take a little rest, after which the symptoms usually disappear. The young, ambitious and less experienced diver however insists on carrying on with heavy work with the obvious risk of losing control of himself. A very frequent symptom is the occurrence of headache during and after the dive. These symptoms are characteristic of exposure to increased CO_2 concentration of 3% and higher (Schaefer 1958, 1962).

When the author discovered by analysis of the air from the exhaust valve that fresh air was to a great extent shunted directly from the inlet to the exhaust valve, he put an airtight metal cap around the exhaust valve and connected a 25 - 30 cm long plastic tube to the cap with the lower perforated end of the tube placed under the breast plate. Under these conditions only the air from the lowest part of the compartment could enter the exhaust valve; striking results were observed, the P_{CO_2} in the inspired air in the helmet was reduced 30-40%, the divers were relieved, experienced no headaches, and less depth narcosis.

The latter finding points to the additional effects of CO_2 and nitrogen narcosis, which are discussed below. The divers also chose on their own volition considerably higher working levels.

CARBON DIOXIDE RETENTION AND HYPERVENTILATION DURING PROLONGED EXPOSURE TO HIGH PRESSURE BREATHING HELIUM-OXYGEN MIXTURES

In preparation for SeaLab operations, experiments were carried out in a dry chamber, in which three subjects were exposed for 12 days to 7 ATA, breathing a gas mixture of 92% helium, 3.5% oxygen and 4.5% nitrogen.

Maximum breathing capacity decreased 38% on the first day of compression and remained at this level for the rest of the exposure period (Lord, Bond and Schaefer, 1966). Tidal volume and alveolar carbon dioxide excretion were significantly increased throughout the exposure, findings which indicate a marked carbon dioxide build-up (Schaefer, Bond, Mazzone, Carey and Dougherty 1968). The carbon dioxide retention found under these conditions is difficult to explain. Several factors must be considered. The increase in breathing resistance was not large enough to account for the carbon dioxide retention. There was no evidence of narcotic effects. The level of atmospheric carbon dioxide in the chamber averaged 1.17% sea level equivalent. The response to prolonged exposure to 1.5% carbon dioxide had been found to be quite different, inasmuch

as both pulmonary and urinary carbon dioxide excretion are reduced below control levels during 24 days of uncompensated respiratory acidosis (Schaefer, Hastings, Carey & Nichols 1963).

Further evidence for carbon dioxide retention in the high-pressure environment was obtained by Hamilton et al (1965) in saturation diving at 650 ft (20 ATA). Two subjects were exposed for 48 hours in a dry chamber in which the carbon dioxide concentration was maintained at or below 1% of an atmosphere. The carbon dioxide response curves measured under these conditions were found to be shifted to the right, while the slopes were not changed. The shift to the right is part of the response observed during adaptation to increased carbon dioxide levels (Schaefer 1949). However, the decrease in slope associated with respiratory adaptation to carbon dioxide is missing in the observed changes under high pressure.

Increased alveolar CO_2 tensions during dives were also found by Jarrett (1966) and Salzano et al (1970), who attributed the rise in CO_2 to decreased ventilation as a result of increased gas density.

An absence of CO_2 retention in oxygen-helium dives to 1000 feet was reported by Overfield et al (1969). Moreover, in saturation excursion dives to 800 and 1000 ft, Schaefer et al (1970) did not see any statistically significant changes of $P_{A_{CO_2}}$ during rest and exercise, although individual variations were quite large. In these experiments, the ambient CO_2 level was kept below measurable levels. In the second dive to 1000 ft, the alveolar CO_2 tension tended to decrease in the two divers. This finding together with the increased bicarbonate excretion in the urine, decrease in chloride excretion, and a rise in urinary pH, was interpreted as reflecting a more alkalotic state due to hyperventilation. In line with these observations were electroencephalographic recordings, which showed in one subject throughout the saturation and decompression periods a consistent decrease in mean frequency and an increase in percent of slow waves, 6-8 Hz (theta activity) correlated with CO_2 excretion in the urine (Proctor et al 1972).

Figure 3 shows the time course in alveolar carbon dioxide tension, urinary CO_2 excretion, and EEG changes during the saturation-excursion dive to 1000 ft and the subsequent two decompression periods. The second decompression period followed a recompression from 30 to 527 ft to treat persistent bends.

The importance of hyperventilation in saturation excursion dives has been emphasized by Brauer (1968) and Bühlmann (1969) who carefully instructed their subjects not to hyperventilate during the dive.

Between 1968 and 1972, the depth of simulated dives in chambers using helium-oxygen mixtures increased to 2001 ft. No evidence of CO_2 retention was found in any of these deep dives under resting conditions. In the British dive to 1500 ft, Morrison et al (1972) observed that the alveolar carbon dioxide partial pressure was slightly lower at rest (= 34 mm Hg) and unchanged or

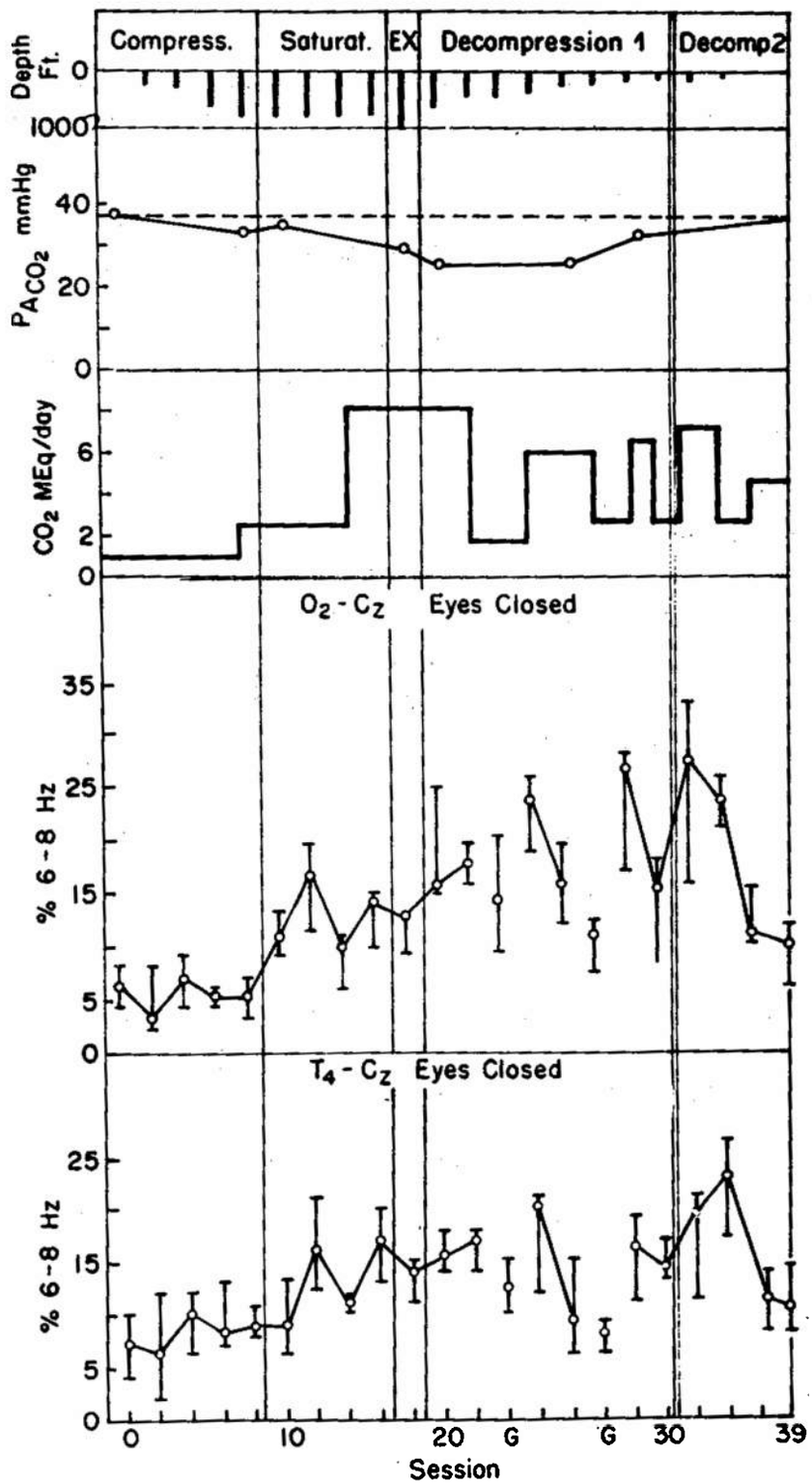

Fig. 3. Subj. C.D. time course in alveolar carbon dioxide tension, urinary CO_2 excretion and EEG changes (percent 6-8 Hz. frequency), two electrode positions $O_2 - C_2$ and $T_4 - C_2$, eyes closed. From Proctor, Carey, Lee, Schaefer, and van den Ende (1972) with permission of the publisher.

increased during moderate exercise of 300 mkg/min. as compared with measurements made at the surface (= 46 mm Hg). Respiratory ventilation was increased both at rest and during exercise, tidal volume increased and respiratory rate declined with depth. Studies of cardiorespiratory functions during dives to 1640 ft breathing helium-oxygen mixtures by Broussolle et al (1973) showed little change in $P_{a\,O_2}$ and P_{aCO_2} at 1640 ft. There was, however, a slight increase in oxygen consumption and carbon dioxide excretion. No signs of CO_2 retention were observed during the dive to 2001 ft (Fructus et al 1972). In the most comprehensive program of investigation of the limits of human tolerance to high pressures, depths of 2000, 3000, 4000 and 5000 ft of sea water were simulated breathing helium, neon, and nitrogen mixtures (Lambertsen 1972). During exposure to helium with oxygen at a pressure equivalent to 1200 ft, no respiratory limitations and signs of CO_2 retention were observed. When crude neon was inhaled at a depth of 1200 ft, the effects of gas density and respiratory resistance as well as respiratory work were equivalent to breathing helium at a depth of 5000 ft. No evidence of CO_2 retention was found under resting conditions. With heavy exercise, respiratory limitations and CO_2 retention were encountered, conditions which are discussed below.

SHALLOW HABITAT AIR DIVING

Considerable efforts have been made in recent years to extend the depth of saturation diving, using air or normoxic nitrogen-oxygen mixtures for working from a shallow undersea habitat. In Project Tektite, subjects were exposed for 2 months to 1.9 atm. of nitrogen (slightly less than 50 ft of seawater) with natural oxygen tension in a habitat in the open sea.

No effects were observed that would limit the ability of normal men to work for prolonged periods at this depth, (Miller and Lambertsen 1971). Subsequently, a 14-day exposure to 4 ATA (corresponding to 100 ft of seawater) of a normoxic nitrogen oxygen mixture was carried out in a chamber which allowed detailed measurements of respiration, and gas exchange (Lambertsen et al 1973). Slight increases in $PaCO_2$ were found during exposure to rest and exercise. Although the workload during exercise was 70% of maximum capacity at 1 ATA, the subjects were able to complete the exercise at increased pressure. However, there was a marked decrease in minute-ventilation during exercise and concurrent increases in $PaCO_2$, which probably were caused by the increase in airway resistance at this pressure. Some CO_2 retention was also indicated in the slight increase in base excess and buffer base during exposure.

The slopes of the CO_2 response curves measured during exposure showed a marked decrease during acute exposure, which was maintained during the chronic exposure of 14 days.

It was concluded that the increased respiratory work due to the increased density of the N_2-O_2 mixture at 4 ATA caused the decrease in the ventilatory response to exercise and CO_2 inhalation, rather than a suppression of chemosensibility.

The slight respiratory acidosis found during the 14-day exposure to 5.2% O_2 in N_2 at pressure equivalent to 100 ft of seawater was associated with hemoconcentration, a loss in plasma volume (-19%) and a reduction in red cell mass (-25%) (Alexander et al and Johnson et al 1973).

Hamilton et al (1973) reported recently about two saturation excursion dives at 90 and 120 ft with descending and ascending excursions. A normoxic nitrogen-oxygen mixture was used. Pulmonary function tests and respiratory gas exchange studies did not show any significant changes.

Performance tests and evoked brain responses demonstrated a definite adaptation to nitrogen narcosis in divers saturated at 90 and 120 ft. Their performance at 200 and 250 ft during excursion dives showed practically no impairment from control levels.

These studies demonstrated the feasibility of long duration excursions to depths of approximately 300 feet using combinations of air and nitrogen-oxygen breathing mixtures.

Moreover, two 30-day studies using compressed air at simulated depths of 50 and 60 ft of seawater (oxygen partial pressures 0.52 ATA and 0.59 ATA, respectively) were recently carried out at the Naval Submarine Medical Research Laboratory without medical complications (Adams 1974). Pulmonary function studies did not show any significant changes. Ventilatory and pulse rate response to exercise loads of 100 and 150 watts increased during the end of the 4-week exposure. Two sets of CO_2 tolerance curves obtained for normoxic and hyperoxic conditions showed both a slight but significant reduction in slope during exposure. These findings were interpreted as indicating an effect of the increased density of gases. Acid-base balance studies carried out during the 60-foot dive produced evidence of a mild respiratory acidosis by the second week of exposure and lasting throughout the experiment. P_{CO_2}, actual bicarbonate and standard bicarbonate of venous blood significantly increased commensurate with a decrease in base excess. Red cell diphosphoglycerate (DPG) concentration was significantly decreased during the phase of the dive (days 9-20) during which shallow excursions were made and also during the second week of the post-dive period.

Hematological studies revealed in both compressed air saturation dives a decline in Hgb, RBC count and calculated hematocrit through the compression period which became statistically significant during the first week of the post decompression period. The fall of these hematological parameters was greater during the second dive at 60 ft (Murray et al 1974). Reticulocyte counts remained stable through the compression phase but increased to values statistically higher than pre-dive values during the second and fourth week post decompression.

It is of interest to compare the 14-day normoxic exposure to 100 ft with the 30-day hyperoxic exposure to 60 ft in regard to CO_2 retention. An acidosis developed under both conditions. There was an increase in base excess in the 4 ATA normoxic experiment, indicating CO_2 retention. A decrease of base

excess in the hyperoxic compressed air exposure suggests the development of a metabolic acidosis. The slopes of the CO_2 response curves decreased under both conditions.

THE ROLE OF CARBON DIOXIDE IN INERT GAS NARCOSIS

If the diver is exposed to compressed air at pressures higher than 99 to 132 ft (4 to 5 ATA), he shows an impairment of performance and certain personality changes which have been referred to as narcotic effects of nitrogen (Behnke, Thomson & Motley 1935). Bean (1950) suggested carbon dioxide retention as the cause of nitrogen narcosis. His conclusion was based on findings in dogs, where an acute rise in alveolar carbon dioxide was associated with rapid compression. The hypothesis of Bean was supported in recent years by Seusing and Drube (1960) who reported increases in alveolar carbon dioxide tension in air and helium dives in the dry chamber. Bühlmann (1961) also favored the carbon dioxide theory of nitrogen narcosis, based on measurements and calculations of breathing resistance during dives to greater depths. However, he avoided further comments on the carbon dioxide theory in a more recent paper (Bühlmann 1963). Evidence against the theory, implicating carbon dioxide as the likely cause of nitrogen narcosis, has been accumulating in recent years. Rashbass (1955) observed impairment of performance in divers under pressure without an associated rise in alveolar carbon dioxide tension. Moreover, Lanphier and Busby (1962) were able to demonstrate that strong subjective effects of nitrogen narcosis were present when both alevolar and arterial carbon dioxide tensions were low. Schaefer (1965a) pointed out that the symptoms of nitrogen narcosis and those of carbon dioxide effects on consciousness are distinctly different. Bennett (1963) produced the most convincing proof that the carbon dioxide theory of nitrogen narcosis is not correct. He exposed anaesthetized cats to nitrogen, helium, and argon under increased pressures and found a depression of evoked response in the presence of nitrogen indicating a narcotic effect while the brain carbon dioxide tension did not change.

In more recent experiments in man, Bennett and Blenkarn (1973) measured arterial blood gases and performance efficiency in 6 subjects during the decompression stages at 286 feet (9.6 ATA) and 190 feet (6.7 ATA) following a saturation-oxygen-helium dive to 870 ft with excursion to 1000 ft. The arithmetic tests scores fell by 34.5% when the subjects were breathing air at 286 feet, but were not different from the control values during breathing of a 20/80 helium-oxygen mixture.

The Pa_{CO_2} values obtained during breathing air or oxygen-helium at both decompression stages did not show any significant differences. They ranged between 31 and 35 mm Hg, indicating a hypocapnia. A positive base excess of 4.1 meq/L was observed which the authors interpreted as a chronic metabolic adaption to a mild hypercapnia of 44-50 mm Hg under steady state conditions. Such a mild degree of hypercapnia had been determined under steady state conditions by Saltzman et al (1971) in three men at 200 feet and 250 feet breathing alternately normoxic oxygen-

nitrogen and normoxic oxygen-helium. The P_a CO_2 was in both cases 45 mm Hg as compared to 40 mm Hg on the surface while breathing air.

When subjects breathed a nitrogen-oxygen mixture, an increase in reaction time and an increased in failures to respond to the reaction time test were observed. During helium-oxygen breathing no changes were found, although the arterial CO_2 level was elevated to the same extent. From both experiments, the conclusion could be drawn that the raised arterial carbon dioxide is not the direct cause of the inert gas narcosis.

Hesser et al (1971) attempted a delineation of the nitrogen and CO_2 component in compressed air narcosis by comparing the effects of inhalation of gas mixtures containing 0, 2, 4, and 6% CO_2 - sea level pressure equivalent in air at 6.0 ATA and in O_2 at 1.3 ATA. The inspired O_2 pressures were approximately 1.2, ATA in both conditions, while the inspired N_2 tension differed by 4.7 ATA. The performance changes measured under both conditions plotted against the alveolar CO_2 tension had no significant effect on the nitrogen component, however the narcotic action of CO_2 was enhanced with increasing N_2 pressure. High alevolar N_2 and CO_2 pressures are additive in their effects on performance; below 40 mm Hg, the CO_2 component is negligible.

This investigation enlarged on the older observations of Case and Haldane (1941) who concluded that CO_2 may not be the cause of, but might well enhance the development of nitrogen narcosis.

Severinghaus (1974) recently discussed the problem of CO_2 narcosis on the basis of data obtained in experiments in which the anesthetic potency of CO_2 was determined by titrating CO_2 against N_2O in volunteers (McAleavy et al 1961). The endpoint of performing a coordinated task, which coincided with falling asleep, was first determined by raising slowly the N_2O concentration. Then the P_{CO_2} was varied in the subjects. After reaching a steady state of P_{CO_2} of 25, 40 and 55 mm Hg, N_2O was added. The concentration of N_2O was slowly raised until the subjects fell asleep. As can be seen from Figure 4, the effective concentration of N_2O was markedly lower at higher P_{CO_2}. An increase of P_{CO_2} of 10 mm Hg decreased the anesthesia endpoint by 50 mm Hg of N_2O pressure.

The results of these studies demonstrate clearly that the effects of the two anesthetic agents CO_2 and N_2O are additive. According to Severinghaus, this type of analysis is applicable to nitrogen narcosis and has been carried out for nitrogen, helium, and other inert gases. All of them have been found to possess additive anesthetic potencies with the exception of helium. The latter has two effects, an anesthetic effect at enormous pressures of 200 atm, but at this level pressure per se has a stimulating effect.

The CO_2 narcosis effects have been related to the extracellular pH (Eisele et al 1967). The lipid-like anesthetic activity of CO_2 has not been tested because it seems to produce acidotic anesthesia at much lower levels.

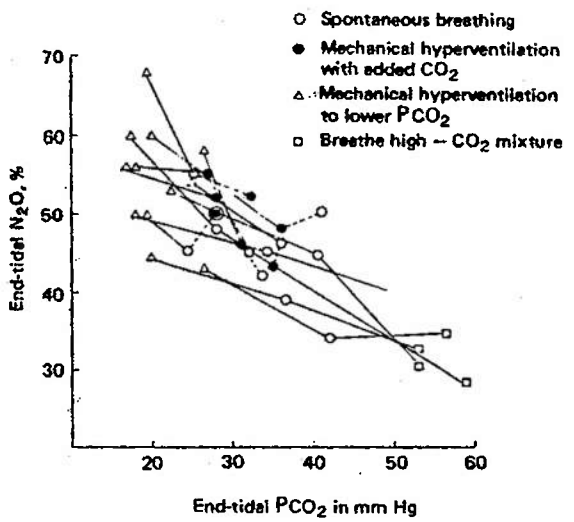

Fig. 4. The anesthetic potency of N_2O is altered by CO_2, such that less N_2O is required to obtain unconsciousness at higher P_{CO_2}, suggesting that CO_2 (or H+) have anesthetic potency five times as great as N_2O

From Severinghaus (1974) with the permission of the publisher

RESPIRATORY RESPONSE TO CO_2 AND THE CONTROL OF VENTILATION AT HIGH PRESSURE

The marked CO_2 retention found in divers under increased pressure breathing of nitrogen-oxygen mixtures by Lanphier (1963), and in divers during exposure to a helium-oxygen-nitrogen mixture at 7 ATA with an ambient CO_2 level of 1.1% by Schaefer et al (1968), have raised the question whether exposure to pressure alters the sensitivity of the respiratory center to carbon dioxide, resulting in a decreased ventilation, or whether the increased work of respiration depth is responsible for the rise in $PaCO_2$ and fall in ventilation.

Wood and Bryan (1970) and (1971) provided quantitative data which indicate that the sensitivity of the respiratory center to CO_2 is not changed under increased pressure. However, the efficiency of the respiratory work is reduced. A CO_2 breathhold time curve and the rebreathing CO_2 ventilatory response curve (Read test) were obtained at 1.0, 4.0, and 7.0 ATA. Moreover, esophageal pressure-volume curves were measured during the Read test to derive inspiratory work. The slope in the breathholding CO_2 response curve did not change, but the ventilatory response to CO_2 decreased at depth. For a given P_{CO_2}, inspiratory work and tidal volume did not change with increased pressure, a fact which has been interpreted as an indication of the unaltered sensitivity to CO_2. Because of the decrease in air flow rates and respiratory frequency at depth the same inspiratory work produces less ventilation at depth. The ventilatory response to CO_2 is therefore diminished. Similar data were obtained by Doell (1973). Using a rebreathing method they examined the ventilatory responses to CO_2 and hypoxia at 1 and 4 ATA. Ventilatory responses to CO_2 were less at 4 ATA than at 1 ATA, as is shown in Figure 5. Since the ventilatory responses to hypoxia were the same at 1 ATA and 4 ATA, at normal Pa_{CO_2}, the conclusion was drawn, that the output of the central chemoreceptors, as shown by the hyperoxic CO_2 response was expressed in inspiratory work, while the output of the peripheral chemoreceptors, as shown in the normocapnic hypoxic response, was reflected in ventilation.

The author noted that three of the subjects used slower frequencies and larger tidal volumes at depth in response to the hypercapnic stimuli resulting in a work conserving breathing pattern.

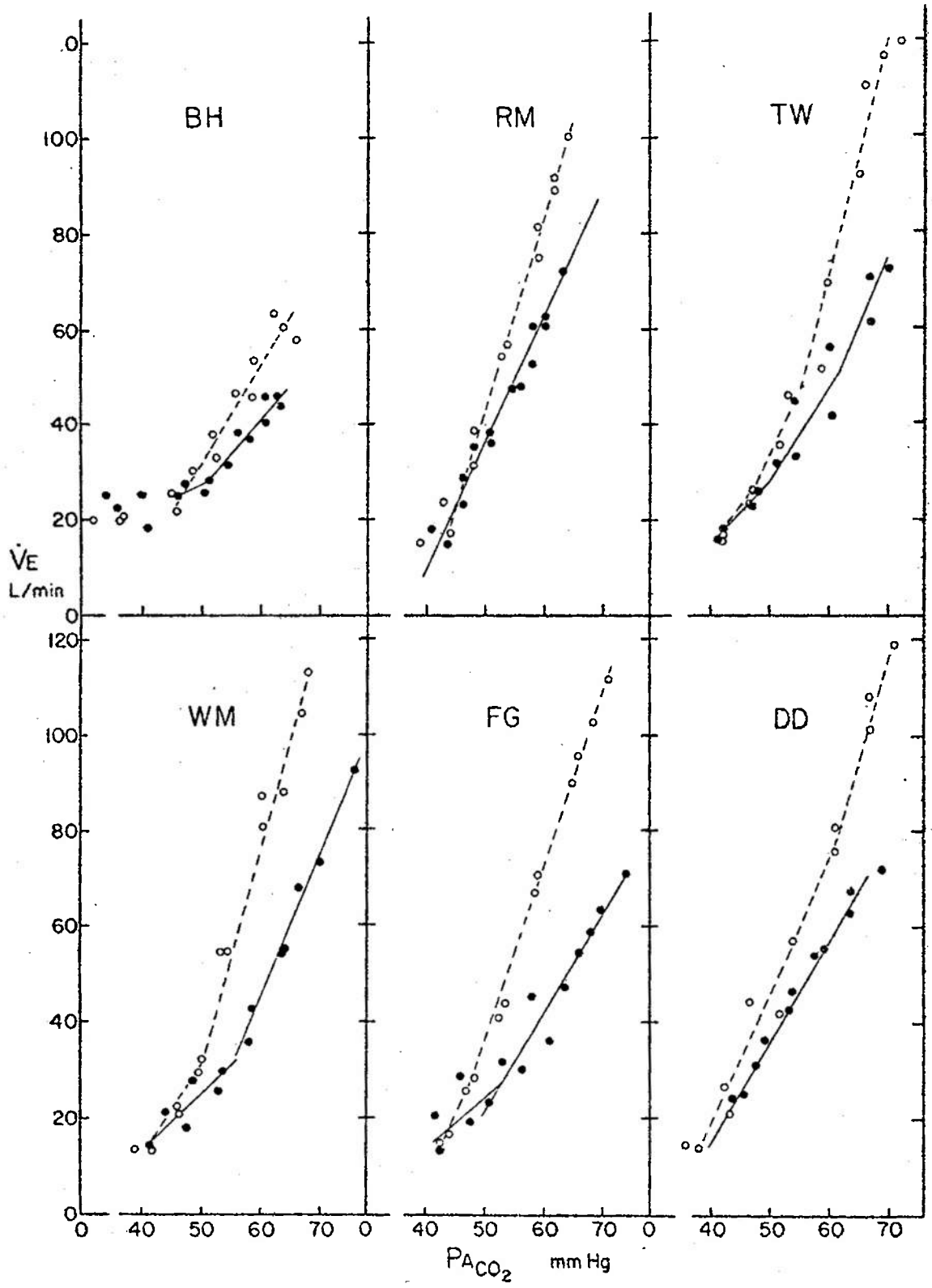

Fig. 5. Hyperoxic CO_2 response curves in 6 subjects ordinates: ventilation in L/min, Abscissae: P_{ACO_2} in mm Hg. Shown for each subject are data from two rebreathing runs at 1 ATA/open circles – dashed line) and two rebreathing runs at ATA (solid circles, solid lines). The curves were fitted by eye. From Doell (1973) with permission from the publisher.

However, no change in the tidal volume - frequency combination was observed in response to the hypoxic stimuli at depth.

Fagraeus and Hesser (1970) also studied the ventilatory response to CO_2 in hyperbaric environments when breathing low concentrations of CO_2 in oxygen at 1.7 ATA and in air at 8.0 ATA. The inspired O_2 and CO_2 pressures were approximately the same in the two conditions, whereas the inspired N_2 pressures differed by 6.3 ATA. The rise in N_2 pressure from 0 to 6.3 ATA caused a nearly 50% reduction in slope with no shift in position of the CO_2 response curve.

In view of the fact that the CO_2 response curves obtained from subjects with added breathing resistance also show only a reduction in slope, the author concluded that the reduction of ventilation found under increased pressure was due to the increase in gas density and breathing resistance.

The ventilatory limitations on heavy physical work at increased ambient pressures is one of the primary factors determining the depth limit in diving operations. Because of the increased density of the inspired gas, the work of breathing is increased, a condition which results in a decrease of alveolar ventilation and CO_2 retention. It is well known that the latter may be hazardous or fatal for a diver. Quite a few studies have been carried out in recent years to determine the depth at which respiratory limitations for moderate and heavy work develop. Figure 6 taken from the paper of Fagraeus and Linnarson (1973) is representative of the findings on CO_2 retention in exercise at high ambient air pressures.

It shows typical time courses of breath-by-breath and tidal P_{CO_2} during submaximal (100 watts) and maximal exercise (400 watts) at 1.0, 3.0 and 6.0 ATA air. No changes in P_{ACO_2} are seen during submaximal exercise, but during maximal exercise CO_2 retention is clearly pronounced at 3.0 ATA air and P_{ACO_2} increases to levels of 56 mm Hg at 6.0 ATA air. These observations are characteristic of, and are in line with previous findings on, end tidal CO_2 during submaximal work breathing helium-oxygen mixtures at 600 feet, Bradley et al (1971), at 800 and 1000 feet Schaefer et al (1971), and at 1640 feet, Broussolle et al (1972).

When higher work loads were used, 1200 kilopond-meters per minute, which corresponded to 80% of the subjects' maximal work capacity, subjects were unable to complete this exercise test at 1200 feet of sea water breathing a neon-helium oxygen mixture which has a density equivalent to that of helium-oxygen at 5000 feet Strauss et al (1972). Using these conditions there was a clearly pronounced CO_2 retention. Occasionally, an increase in alveolar Pa_{CO_2} has been found during light work. Broussolle et al (1968) observed in subjects exercising at 55 watts at 7 ATA in air an increase of Pa_{CO_2} to 52.7 mm Hg from a control level of 35.8 mm Hg at 1 ATA. The subjects might have been some of the trained divers, who retain CO_2 instead of increasing their ventilation sufficiently to meet the demands of the exercise. This form of adaptation to CO_2 is discussed below in more detail.

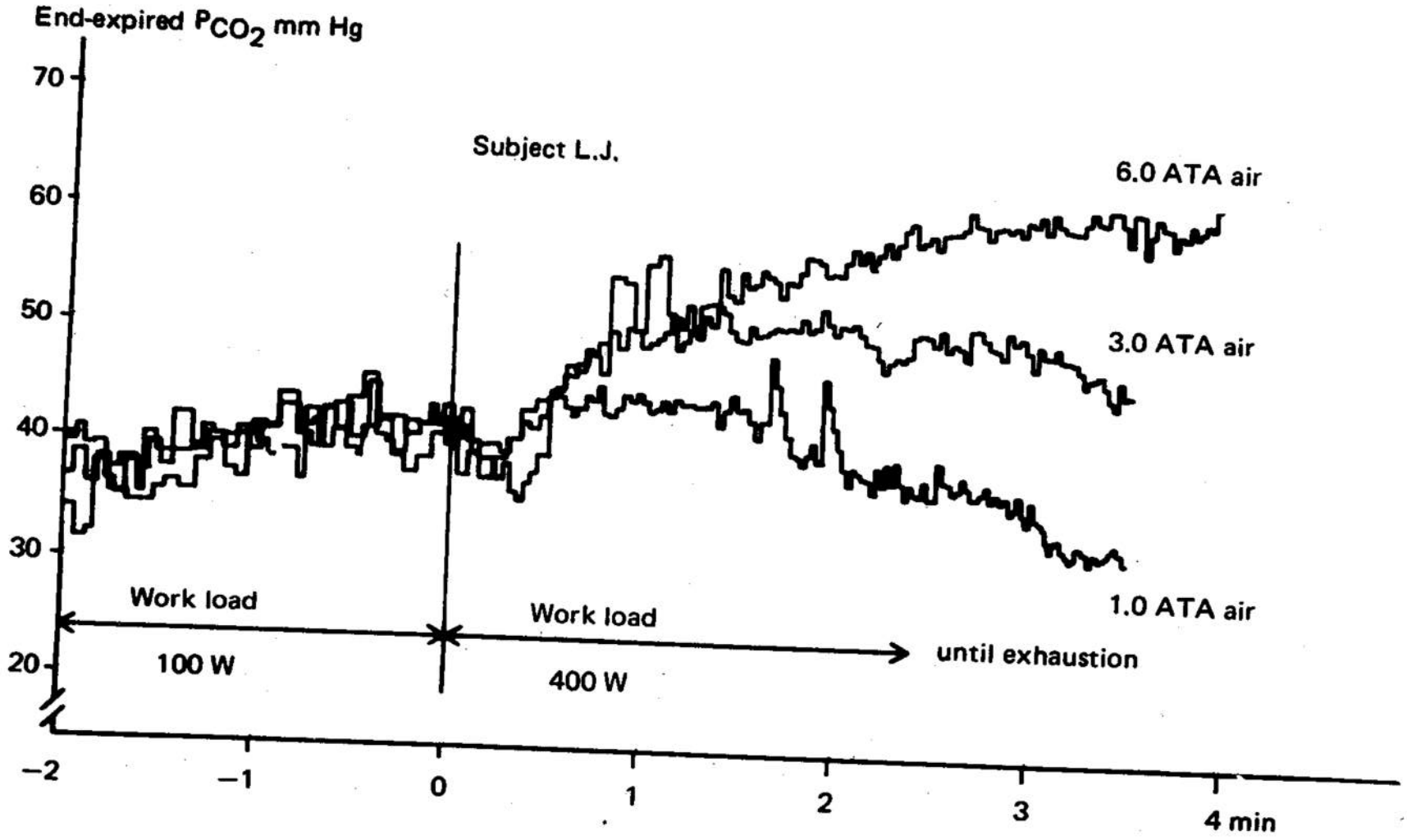

Fig. 6. Typical time course of breath-by-breath and expired P_{CO_2} during submaximal and maximal exercise at 1.0, 3.0 and 6.0 ATA air. From Fagraeus and Linnarson (1973) with permission of the publisher.

Studies of acid-base balance in arterialized blood during heavy work, 1100 kgm/min at 1 ATA and 5 ATA air, showed an increase of $^{Pa}CO_2$ to 70 mm Hg and a decrease of pH to 7.29 at 5 ATA (Kurenkow 1973).

Eklund et al (1973) made an attempt to differentiate between metabolic factors (acidosis) and ventilatory factors (decreased working capacity of the breathing muscles) as possible causes of the respiratory limitations observed during heavy workloads at increased pressures. They compared the effect of CO_2 inhalation (20 mm Hg) on maximal exercise of leg work at an ambient pressure of 2 ATA of a 100% O_2 atmosphere with the effect of increased CO_2 at 2 ATA during maximal forearm work. The latter condition involved a much smaller muscle mass, one that would not lead to great demands on oxygen uptake and ventilation, while in the former condition, the oxygen and ventilation requirements might be limiting.

During maximal leg work and CO_2 inhalation, the arterial P_{CO_2} rose to 55 mm Hg and the venous P_{CO_2} of the femoral vein to nearly 100 mm Hg, in both cases these values were about 10 mm Hg higher than in the condition without CO_2 inhalation; ventilation was about 10 l/min higher during exercise with CO_2 and the performance time significantly reduced (3.4 minutes as compared with 4.7 minutes).

During forearm work, CO_2 breathing produced similar changes in venous P_{CO_2} (99 mm Hg), as compared to 90 mm Hg during controls; however the venous pH fell only to 7.13 as compared to 6.99 in the leg exercise. The performance time for forearm work was not shortened. It was concluded that conditions studied the increased effort of breathing was more important as limiting factor for performance than the induced change in tissue pH.

The venous pH data obtained under both conditions are not really comparable. During leg work the venous pH dropped to 6.99, where enzyme inhibitions occur (Schaefer, 1974), while during forearm work the pH was above this level. However, these conclusions are supported by the results of maximal and submaximal exercise studies during acute and chronic hypercapnia.

Because of the interaction of hypercapnia and exercise, they also apply to exercise at increased pressures and the concomitant increase of $^{Pa}CO_2$. The studies of Luft et al (1974) on maximal exercise at an ambient CO_2 level of 15 mm Hg (which is the highest acceptable limit for emergencies in spacecraft) clearly demonstrated that the respiratory discharge of CO_2 is impeded. A substantial rise in arterial $^{P}CO_2$ during maximal exercise breathing air with CO_2 admixture was observed while the $^{Pa}CO_2$ was reduced during maximal exercise under control condition. Figure 7 shows the acid-base changes during and after maximal exercise with and without 15 mm Hg $^{PI}CO_2$, at the end of the exhaustive work $^{Pa}CO_2$ fell to 30 mm Hg under control conditions, but rose to 41 mm Hg in the last minute of exercise. The time course of $^{Pa}CO_2$ during CO_2 exposure and during recovery was also different. As a result of this rise in $^{Pa}CO_2$ the metabolic acidosis generated by the anaerobic processes in the muscles can no longer be regulated by respiration.

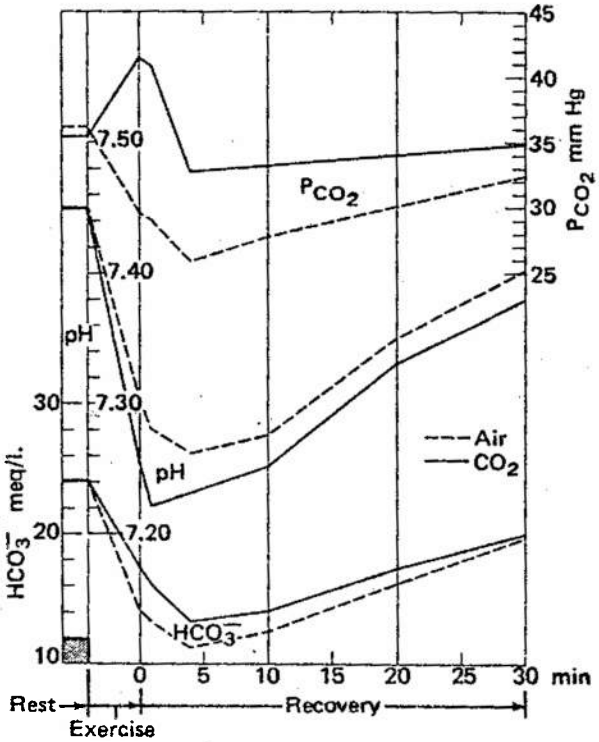

Fig. 7. Acid-base changes during and after exercise with and without 15 mm Hg P_{ICO_2}. From Luft, Finkelstein and Elliott (1974) with permission of the publisher.

It was noted, that the subjects who were pedaling at 50 rpm also were breathing at this rate, when they were close to the maximum minute ventilation of 150 1/min. With the onset of symptoms of dyspnea, some attempted to raise the respiratory rate above the pedaling rate which led to a fall in tidal volume and alveolar ventilation. This is clearly an example of a circumstance where the work of breathing becomes the limiting factor. Symptoms of intercostal muscle pain and subjective distress from the ventilatory effort were reported by subjects performing maximal exercise in 21 mm Hg P_{CO_2} (Menn et al 1970). In both studies, CO_2 elimination was greatly reduced. Similar findings were obtained by Sinclair et al (1971).

Older studies of submaximal exercise during acute and chronic exposure to 3% CO_2 (21 mm Hg) also demonstrated a reduction in CO_2 output and ventilatory efficiency (Häbisch 1949) (Schaefer 1949).

Lanphier (1969) and his coworkers Miller et al (1971) suggested the use of the measurement of maximal voluntary ventilation (MVV) at pressure to predict the exercise level at which ventilation becomes insufficient and CO_2 begins to accumulate. They concluded that a workload at pressure which produces a ventilation corresponding to 100% of MVV is possible.

Shephard (1967) however found that the highest ventilation subjects were able to maintain during near-maximal exercise at sea level was about 70 - 80% of their 15 sec. MVV. Freedman (1970) reported that ventilation at maximal tolerable exercise corresponded to 64% of the 15 sec. MVV. Fagraeus and Linnarson (1973) suggested on the basis of their studies of maximal exercise at 6 ATA air, the use of an exercise load of 60% of the 15 sec. MVV.

ADAPTATION TO DIVING

Carbon dioxide tolerance curves were obtained by exposing subjects for 15 min to 3.3, 5.4 and 7.5% carbon dioxide. Alveolar ventilation and alveolar gas tensions were determined at the end of each exposure period. The carbon dioxide tolerance curves of experienced tank instructors showed a shift to the right and a decreased slope when compared with those of the laboratory personnel (Schaefer 1965 b). As shown in carbon dioxide sensitivity tests

by eight tank instructors, the high tolerance to carbon dioxide is developed during regular diving and is lost after 3 months without diving. The ventilatory response to 5% carbon dioxide is significantly larger at the end of the 3-month layoff period. The change in lung volumes, that is, an increase in total lung capacity, vital capacity, tidal volume, and a decrease in residual volume, may contribute to the reduced sensitivity to carbon dioxide because of the relationship found between large tidal volume, low respiratory rate and low response to carbon dioxide (Schaefer 1968).

Adaptation to breathhold and scuba diving involves an adaptation to carbon dioxide. Adaptation to breathhold diving has been observed in instructors at the Escape Training Tank at the Submarine Base, Groton, Connecticut (Schaefer 1965b).

Other parameters of adaptation to carbon dioxide have been previously established in human subjects during prolonged exposure to 1.5% carbon dioxide. They include besides changes in acid base equilibria, an increase in red cell sodium and a decrease in red cell potassium (Schaefer 1964). Tables 2 and 3 show the distribution of carbon dioxide in plasma and cells and the distribution of electrolytes in plasma and red cells of 11 tank instructors after periods with and without heavy underwater work. It can be seen that after a period of intensive diving, the pH is decreased, the carbon dioxide tension and bicarbonate levels are increased, and the sodium and potassium concentrations in red cells exhibit the typical changes observed in prolonged exposure to carbon dioxide. This data provides further support for an adaptation to carbon dioxide during breathhold diving.

Evidence of an increase in carbon dioxide stores as a result of diving was obtained in instructors, following a 2-year period of diving when compared with data obtained after a 3-month period of no diving. During constant hyperventilation, lasting one hour, more carbon dioxide was eliminated and the end tidal carbon dioxide tension was significantly elevated in the first condition (Dougherty & Schaefer 1962).

The adaptation to carbon dioxide in breathhold diving is associated with an adaptation to low oxygen. Experiments with divers breathing low oxygen mixtures demonstrate that they show a lower ventilatory response and are able to utilize oxygen better than non-divers (Schaefer 1965a).

During skin diving, carbon dioxide intoxication does not appear to be a major problem and is certainly less likely to occur than in scuba diving. The alveolar and blood carbon dioxide levels are rather low throughout a dive and can easily be controlled by regulating the speed of ascent. Moreover, a lowered sensitivity to carbon dioxide, as a result of adaptation, gives divers an additional protection against the acute toxic effects of carbon dioxide.

The high tolerance to CO_2 has been found to be associated with a reduced autonomic response as indicated in smaller elevation of pulse rate and blood sugar during CO_2 exposure. Blood pressure response to an injection

TABLE 2

Effect of daily breath-hold dives during a 6-month period on distribution of carbon dioxide in plasma and red cells (venous blood) of 11 tank instructors

Plasma				*Red cells*	
HCO_3 mmoles/L	H_2CO_3 mmoles/L	pH	PCO_2 mm Hg	HCO_3 mmoles/L	H_2CO_3 mmoles/L
After a 5-month period without water work (control)					
25·1 ± 1·5	1·34 ± 0·09	7·38 ± 0·01	44·7 ± 2·96	16·76 ± 0·87	1·12 ± 0·07
After a 6-month period with heavy water work					
28·3* ± 1·38	1·58* ± 0·18	7·35* ± 0·05	52·7* ± 6·1	18·60* ± 0·67	1·32 ± 0·15

* Differences from controls statistically significant at the 5% level.

TABLE 3

Effect of daily breath-hold dives for a period of 6-months on red cell and plasma electrolytes (venous blood) (11 subjects)

Measured values									*Calculated values*			
Whole blood					*Plasma*				*Red cells*			
H_2O, g/L	Na, mEq/L	K, mEq/L	Cl, mEq/L	Haemato-crit	H_2O g/L	Na, mEq/L	K, mEq/L	Cl, mEq/L	H_2O, g/L	Na, mEq/L	K, mEq/L	Cl, mEq/L
After a 5-month period without water work (control)												
824 ± 12·8	86·8 ± 5·0	43·9 ± 2·3	84·5 ± 2·3	43·0 ± 1·9	924 ± 7·3	142 ± 3·5	4·78 ± 0·62	103·5 ± 3·5	692 ± 27	13·7 ± 3·8	95·8 ± 15·4	59·2 ± 6·6
After a 6-month period with heavy water work												
811* ± 5	86·8 ± 2·9	34·6* ± 2·4	83·5 ± 2·4	44·8 ± 2·3	915† ± 9·3	133* ± 4·4	4·09† ± 0·39	103·5 ± 1·6	679 20	30·4† ± 17·3	72·1* ± 5·4	59·4 ± 6·9

* Differences from controls statistically significant at the 1% level and better.
† Differences from controls statistically significant at the 5% level.

of a cholinergic drug, Mecholyl, was measured in a group of 13 divers and 19 laboratory personnel (Schaefer 1965). The divers exhibited a significantly smaller fall in blood pressure than the group of laboratory personnel. These findings suggest that the adaptation to breathhold diving produces a damping effect on the cholinergic system. The stress resistance found in divers is in the line with their subjective observations of increased "relaxation" in the course of prolonged diving training.

Song et al (1963) found that the diving women of Korea has a lower ventilatory response to CO_2 than non-diving women, however their response to hypoxia was not different.

In the majority of studies in scuba divers, adaptation to CO_2 was observed with the exception of an investigation by Froeb (1961). He compared the respiratory response to carbon dioxide in 16 professional divers using scuba equipment with those of non-divers and did not find any evidence of adaptation to carbon dioxide in the scuba divers. In studies of well-trained underwater swimmers of the U. S. Navy and untrained swimmers (laboratory personnel) using a closed-circuit oxygen breathing unit, a higher mean end tidal carbon dioxide tension was found in the trained swimmers during swims at a speed of 1.1 to 1.8 km/hour (Goff & Bartlett 1957; Goff, Brubach & Specht 1957). During resting conditions under water, differences in end tidal carbon dioxide tensions were negligible. Findings indicated some degree of adaptation to carbon dioxide in the trained swimmers which perhaps in part, was related to the noted respiratory pattern with long post-inspiratory pauses. Furthermore, adaptation to increased work of the inspiratory muscles might have contributed to the elevated carbon dioxide tensions in the trained underwater swimmer because it was shown that alveolar carbon dioxide tensions increase linearly with the work-load on the inspiratory muscles (Milic-Emili & Tyler 1962).

Lanphier (1963) found in a group of scuba divers end tidal P_{CO_2} values of 50 mm Hg during moderate exercise under water at 4 ATA, breathing air. Some of the "CO_2 retainers" of this group were subsequently studied in more detail and found to show large increases in $P_{A_{CO_2}}$ during moderate exercise in air at normal atmospheric pressure, although they used a low resistance breathing apparatus.

Broussolle et al (1968) measured the ventilatory response to increased CO_2 concentrations in scuba divers and found a reduction of 30% compared to non-divers. Broussolle et al (1972) also observed in trained scuba divers

a marked reduction in the ventilatory response to different workloads (55, 110 and 165 watts) associated with higher alveolar CO_2 values in comparison to non-divers. The CO_2 elimination in the trained SCUBA divers was less, particularly at higher workloads (110 watts). A marked reduction in the CO_2 response of trained divers was also found by Varene et al (1972).

Recent studies of exercise hyperpnea in divers, non-divers, and runners by Lally et al (1974) showed significant lower steady state ventilatory responses in divers as compared to controls (laboratory personnel) in exercise, walking at 10% grade at 1.2 and 3 mph. Moreover, the fast (neural) component of the initial 15 sec. ventilatory response was also less in divers. The values for runners were intermediate between divers and sedentary non-divers (Figure 8). Both divers and athletes had higher $P_{A_{CO_2}}$ values and lower RQ values than the sedentary control group, although divers had higher values than athletes. End tidal $P_{A_{CO_2}}$ was about 5 mm Hg higher in divers at different levels of exercises, as compared to those of sedentary non-divers. Divers showed a markedly slower and deeper breathing pattern than the other two groups. It is concluded that this unusual ventilatory behavior is not fitness-related and may involve besides a reduced chemosensitivity a conditioned response phenomenon.

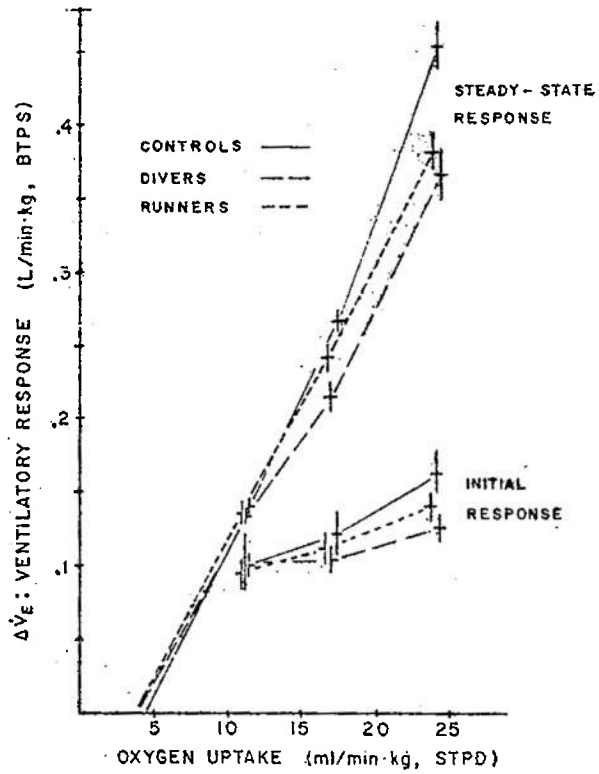

Fig. 8. Steady state and initial (neurogenic) ventilatory responses to exercise of the three groups of subjects as a function of VO_2. From Lally, Zechman and Tracy (1973) with permission of the publisher

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13. ABSTRACT

Exposure to the increased barometric pressure, such as encountered under water, affects the mechanics of respiration and in particular the behavior of respiratory gases. Carbon dioxide plays a major role in the physiology of the high-pressure environment since the increased breathing resistance easily leads to carbon dioxide retention. The latter has been frequently noted in scuba and helmet diving. Pulmonary gas exchange in breathhold diving is influenced by the compression and decompression events, resulting in a reversed carbon dioxide gradient during descent. The alveolar carbon dioxide level can be controlled during the ascent from depth by controlling the speed of ascent which is of significance to both breathhold diving and the buoyant ascents of submarine escape. Investigations of pulmonary gas exchange in rest and exercise during exposure to high pressure while breathing helium-oxygen gas mixtures have demonstrated the existence of respiratory limitations and associated CO_2 retention when divers are performing heavy work. In shallow habitat air diving using combinations of air and normoxic nitrogen-oxygen breathing mixtures, evidence for the development of slight respiratory acidosis and CO_2 retention was obtained.

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Breathhold diving						
Submarine escape (free and buoyant ascent)						
Self-contained diving						
Helmeted diving						
Carbon dioxide retention and hyperventilation during prolonged exposure to high pressure breathing helium-oxygen mixtures						
Shallow habitat air diving						
Role of carbon dioxide in inert gas narcosis						
Respiratory response to CO_2 and the control of ventilation at high pressure						
Adaptation to diving						